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EDITORIAL.

Our Graduating Degree

The graduate in Medicine from the University of Toronto has long had to be content with the degree of Bachelor of Medicine, in accordance with the degree-granting custom of many of the Universities of England and Scotland. However it is the custom of this continent to grant the Medical graduate the degree of Doctor of Medicine, and in many cases an additional degree, Master of Surgery.

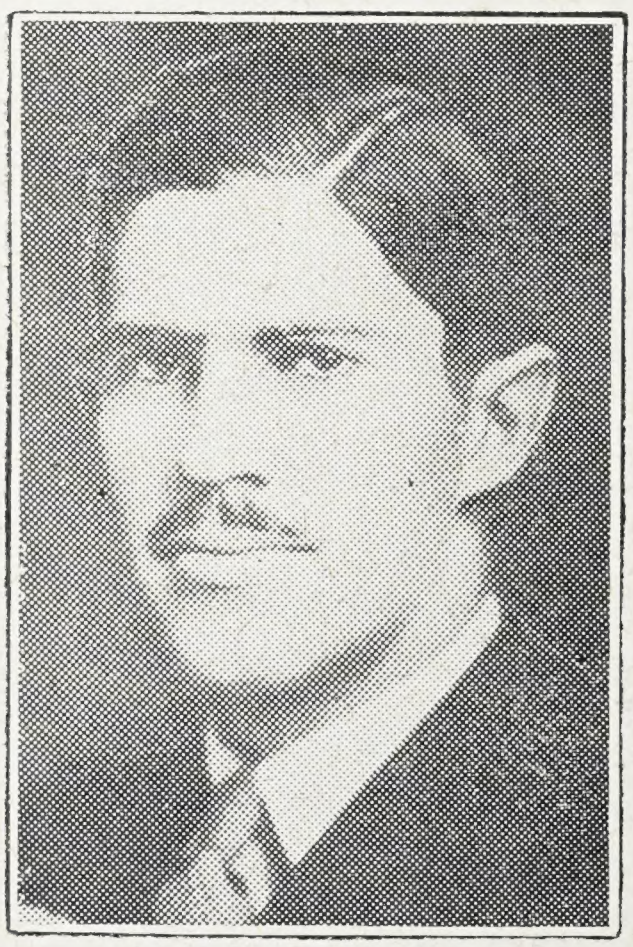
There is much to be said for the old country system of granting degrees. The term doctor, which to the laity signifies a medical man in academic circles retains much of its original meaning, viz., teacher, from the Latin "doceo," meaning to teach. In other lines of academic endeavour besides Medicine, no man is entitled to use the term doctor until he has completed a very extensive course beyond the requirements of undergraduate study, involving much original work, thus demonstrating his capacity to advance the particular branch of learning in which he is interested, and giving him the right to be considered as a teacher by those studying his particular science. In accordance with this ideal, the University of Toronto has reserved the degree of Doctor of Medicine for those who are willing to spend three years in specialized clinical or laboratory research after they have completed their undergraduate course.

But to the laity all medical men are doctors, and even here in this university, all who hold the M.B. degree are addressed as "Doctor." Dictionaries, in accordance with common usage define doctor first of all as a practitioner of Medicine, and secondly as a teacher. The average layman, on this continent at least, considers the term M.D. as synonomous with "Practitioner of Medicine." The graduate of our university is often placed in an invidious position when several times in the course of a week he is called upon to explain why his shingle is inscribed, "John Smith, M.B.", while up the street is another shingle bearing the legend "Peter Jones, M.D., C.M." He has to explain that he, John Smith, has taken just as extensive a course as Peter Jones, and ought to be just as competent a practitioner. The layman is not usually in a frame of mind to appreciate just the significance of the high ideals of the University of Toronto, and in spite of a voluble exposition of the case by John Smith is apt to consider that Peter Jones must be the better man because he is a real honest-to-goodness M.D., and has a couple of extra letters besides.

Another case of confusion arising from our plain unvarnished "M.B." has just been brought to our attention by a recent honor graduate from this university who has been doing post-graduate work in Britain. This man was applying for a position with one of the great teaching hospitals of the old country. They replied to his application saying that they understood that he had a degree in Medicine, but wanted to know what were his surgical qualifications. He wrote back and explained. The hospital replied that they could not accept a man without surgical qualifications. In a flash of inspiration our graduate wrote back saying that he had passed the examinations of the College of Physicians *and Surgeons* of the Province of Ontario, and was at once accepted, seeing that he had the necessary surgical qualification.

In view of the fact that most of our graduates go forth to practise medicine in this continent where the vast majority of medical men are M.D.'s, where the great untutored laity do not appreciate the fact that M.B. (Tor.) means more than M.D., C.M. (any other university) we think that our graduates now in practise, and the undergraduate body of this university will welcome the change that is now being contemplated by our senate, viz. the granting of the degree of Doctor of Medicine to all graduates in Medicine of the University of Toronto.

In Memoriam.



DR. KENNETH ARTHUR FIDLER

Dr. Kenneth A. Fidler, a member of the 1927 Graduating Class of the University of Toronto passed away on February the 16th after a five-day illness caused by pneumonia.

He was born in Brockville and lived there with his parents until eight years of age, when the family moved to Woodstock, then to Hamilton, and finally to Toronto where he has resided since that time.

Taking a prominent part in every activity in which he participated, Dr. Fidler was an all-round student in his undergraduate years and gave promise of a successful and brilliant career. He took a keen interest in all athletics and was Intercollegiate Boxing Champion in his weight as well as a member of the Senior Medical rugby team.

In his 26th year, and endowed with a cheery disposition, a straight-forward and alert mind, Dr. Fidler's untimely death was a great shock to his many University associates and friends throughout the city. He was a highly esteemed member of the Kappa Sigma Fraternity and at the time of his death was an interne in the Toronto General Hospital.

Paretic Neurosyphilis.

(By C. M. Crawford, B.A., M.D., C.M.),

Assistant Medical Director Toronto Psychiatric Hospital

Synonyms: General Paralysis, Dementia Paralytica, General Paresis of the Insane (G.P.I.), Parenchymatous Neurosyphilis.

General Paresis, or to use a more truly descriptive term, Paretic Neurosyphilis, is a toxic-organic disease of the central nervous system, caused by the Treponema Pallidum and manifested by certain very definite neurological signs and by a gradual mental deterioration. Superimposed upon various personality types which in all probability determine the mental reactions, it is described as presenting the following clinical patterns: manic; depressive; agitated; simple dementing; paranoid; neurasthenic; and schizophrenic.

History.

The credit for the first fairly accurate description of General Paresis should go to the English pharmacist, Haslam, of the Royal Bethlem Hospital, London, (Bedlam), who, in 1798, wrote a creditable monograph describing the disease. in 1822 A. L. Bayle, a French clinician, more clearly differentiated the condition. The first complete description of the disease entity was written by Calmeil in 1826, under the caption "De la Paralysic Considérée chez les Aliénés." Esquirol first described the characteristic speech defect and Baillarger in 1846 first used the term Dementia Paralytica. Sandras, in 1852, suggested syphilis as one of the principal causes of the disease, and in 1857 Esmarch and Jessen, Danish investigators, agreed with this opinion. On the other hand, Charcot, Dé Jerine and Binswanger denied that there was any relation between the two diseases. Lumbar puncture introduced by Quincke in 1890 led the way to the demonstration of definite changes in the C.S.F. of paretics. The isolation of the spirocheta pallida by Schaudinn in 1905 and the application in 1906 by Wassermann, Neisser and Bruck of the Bordet-Gengou complement fixation test to the blood and C. S. F. of paretics practically proved the relationship between syphilis and General Paresis. The final link in the chain was forged when Moore and Noguchi in 1913 found the spirocheta pallida in the cortex of paretics.

Etiology.

The sine qua non of General Paresis is syphilis. Less than 5 per cent. of syphilitics later develop General Paresis. To explain this many theories are suggested:

1. French neurologists have postulated a *neurotropic* strain of the spirochete with an affinity for the nervous system, as contrasted with the *dermatotropic type* which tends to produce florid skin symptoms. They instance in proof of their contention the occasional cases of conjugal paresis and the fact of several syphilitics later developing paresis when the original infection can be traced to a common source.

2. It is suggested that individuals who later develop Paresis have a nervous system, which has diminished resistance to that particular infection. In other words, it is more-or-less a matter of immunity reactions.

3. It is further suggested that General Paresis develops only in those individuals who, had they not been infected with syphilis, would later have developed some other type of mental disorder. The type of the mental disorder is only conditioned by the original syphilitic infection.

The period of incubation runs between five and thirty years, the average being about ten to twelve years. Males

are more frequently affected than females in the proportion of three or four to one. It is thought that the increased activity of the endocrine glands and the hypercholesterolaemia of pregnancy explain to a certain degree the protection afforded women. Single and nulliparous women present the same frequency as men. Between thirty-five and forty-five appears to be the commonest decade during which the disease appears. Bleuler thinks that alcoholism is a definite predisposing factor. Other clinicians mention as percipitating factors various infections, influenza, emotional stresses and cerebral trauma. It is also claimed that the original luetic infection in the vast majority of individuals who later develop paresis is of a mild nature and often such as to escape detection and proper treatment. A rather interesting point in connection with lues in general is that in the secondary stage about 25 per cent. of cases show a lymphocytosis and increased globulin and protein content of the C.S.F. It is thought that certain of these cases are the ones which later develop General Paresis. This suggestion would emphasize the great importance of an examination of the C.S.F. during the course of treatment of systemic syphilis. General Paresis constitutes from 10 to 20 per cent. of all first admissions to state hospitals in the United States. It is much more common in urban than in suburban communities. Soldiers, sailors, commercial travellers, railway men and hotel employees probably because of their tendency towards irregular sexual habits are especially predisposed to the disease.

Pathology.

The pathological findings in General Paresis are fairly definite. So much so that the condition can be quite definitely diagnosed as General Paresis at autopsy. There is very often a general reduction in the total weight of the brain. The convolutions, especially of the frontal and parietal portions of the brain, are frequently somewhat trophied and the sulci are correspondingly widened. The dura is frequently adherent here and there to the calvarium. In places also there are patches of pachymeningitis hemorrhagica both externa and interna. One frequently also sees apparent opacities of the meninges in spots. The pia mater is also frequently adherent to the subjacent cortex so that when one attempts to strip the pia from the cortex pieces of the cortex are torn away (decortication). Granular ependymitis is frequently seen in the floors of the ventricles and is especially marked in the neighborhood of the choroid plexuses. The ventricles are frequently widely dilated, with an increase of cerebrospinal fluid.

Microscopically there are a number of rather characteristic findings. The regular arrangement of the ganglion cells in layers is frequently altered. The adventitial sheaths of the smaller blood vessels are infiltrated with lymphocytes and plasma cells. This infiltration frequently extends out into the neighboring ganglion cell areas, resulting in more or less ganglionic degeneration. Various degrees of ganglion cell destruction are observable, such as sclerosis, eccentricity of the nucleus, vacuolization and granular degeneration. In addition to this ganglion cell destruction there is a marked proliferation of the neuroglia. Associated with this proliferation there is the appearance of rod cells and spider cells. Satellitosis and neuronophagia are to be found quite commonly, indicating a further tendency towards ganglion cell destruction. There is increased vascularity with the formation of new capillaries.

Symptomatology.

The symptoms of the disease may be classified into—

(a) Neurological and Physical.
(b) Serological.
(c) Mental.

(a) Neurological and Physical:

1. **Pupillary Changes.**—The most common and distinctive symptoms are to be found in connection with the pupillary reactions. These are also some

of the earliest symptoms. The most common condition here is a sluggish reaction of the pupil to direct light as well as a sluggish consensual light reaction. A total absence of light reaction (Argyll-Robertson Pupil) in my experience is not common, although its frequency has been reported to be as high as 45 per cent. of cases. Some interference in the normal light reaction of the pupil is present in at least 75 per cent. of cases. Inequality of the two pupils (anisocoria) is quite common. Frequently the pupils are not concentric, presenting oval outlines or showing notching. In taboparesis both pupils are frequently quite contracted and unequal. Loss of the sympathetic light reflex on one or both sides is reported as being present in about 64 per cent. of cases.

2. **Tendon Reflexes.**—The commonest finding probably absent knee jerks, especially in the tabo-paretic type. Also quite common is the presence of exaggarated and unequal tendon reflexes, explainable upon the basis of removal of inhibition on the part of the controlling cortex.

3. **Muscular Symptoms.**—Some inco-ordination of both upper and lower extremities is rather common. Practically always present is some inco-ordination of the muscles of speech, resulting in the so-called slurring speech (anarthria and dysarthria). This is brought out by the use of test phrases such as "truly rural," "Methodist Episcopal," "British Constitution," "Imperturbability," "Hippopotamus," etc. The facial muscles of expression also tend to lose their tone resulting in a more-or-less ironing out of one or both sides of the face and obliteration of the naso-labial folds. Hand-writing defects are especially common, manifested by dropping syllables, repeating letters, variation in the strength of the strokes and a general tremulousness of the script. The gait is frequently disturbed and is described as being more-or-less of an ataxic type. There is a tendency for the person to walk as if slightly intoxicated. Tremors of various muscle groups are common. This is especially noticeable in the tongue. When it is protruded it is jerked back and forth constituting what is described as the "trombone" tongue. Tremors of the facial muscles are also quite common, brought out by asking the patient to show his teeth. Digital tremors are frequently present also, elicited by asking the patient to stretch out his hand with fingers separated.

4. Occasionally the Babinski sign is present as well as the Gordon and Oppenheim signs. Rhombergism is frequently present in these cases.

5. **Accessory Physical Symptoms.**—Under this heading should be mentioned the tendency towards various types of seizures, such as syncopal attacks, epileptiform or apoplectiform attacks. Sometimes the disease is ushered in by one of these attacks. They are almost invariably of short duration and the patient frequently regains consciousness in a few hours with apparently very little organic damage left. Another rather common late result of the paretic process is osteoporosis or decalcification of the bones. As a result of this the paretic is very liable to sustain bone fractures from very little violence. This is an important point to keep in mind in the care of paretics. There is also frequently a hypalgesia and anaesthesia of the skin as a result of which paretics sustain burns and scalds without complaining. Hence one should be very careful in the application of heat to such patients to see that it is not extreme. A late symptom is incontinence of urine and faeces which makes the care of patients very trying. Decubitus (bedsores), muscle contractures and persistent grinding of the teeth are common symptoms of the terminal stages.

(*b*) *Serological*:

In practically all cases of General Paresis the blood Wassermann is strongly positive. The frequency of this symptom is given as about 99.5%

of cases. In at least 98% of cases the C.S.F. also presents a strongly positive reaction in minimal quantities, viz. .2 c.c. There is generally an increase in the cell count. Practically anything over ten lymphocytes in the miscoscopic field is suggestive. The globulin and protein content of the C.S.F. is markedly increased. The butyric acid test of Noguchi is probably the most satisfactory one to use. The colloidal gold test gives a very characteristic curve in cases of General Paresis, called the paretic curve. The maximum precipitation occurs in the first four or five tubes with a lessened precipitation in the next two or three and none in the last two or three. The test should read, therefore 5555431000. When lumbar puncture is done the C.S.F. as a rule gushes out of the needle. It is, as a rule, clear, with a few flakes floating in it. One text book describes it as occasionally clouded, but I do not think that it is ever so in cases of uncomplicated paresis. Use of the spinal manometer shows the C.S.F. to be under considerably increased pressure.

(*c*) *Mental*:

As a rule the prominent mental symptoms are as follows:

(1) A gradual change of character always for the worse. This is evidenced by the individual committing acts which one would not expect him to commit in view of his previous respectable character. A previously respectable member of the community will start to keep company with individuals of low character or will commit various misdemeanors such as issuing cheques without there being any funds or other petty crimes.

(2) Another very suggestive symptom is due to the patient's temporary disorientation for place. Occasionally the individual will go down town and be unable to find his way back home although he has lived in the district all his life.

(3) There is frequently a gradually increasing lack of care of the personal appearance. The individual becomes more and more untidy about his clothing and will go around with vest or trousers unbuttoned and cravat not properly tied.

(4) Loss of memory especially for recent events is rather common.

(5) Notwithstanding the quite definite mental deterioration the individual as a rule does not recognize that there is the slightest thing wrong with him and expresses himself as "feeling fine."

(6) Marked emotional variability is common, the patient becoming unduly irritable with his own family, while at other times showing excessive good humor, which is out of all keeping with the circumstances

(7) Another rather common symptom is an increased fatigability. The patient complains at times of being very easily tired out.

(8) Frequently at this time also there develop delusions of a more or less absurd type, either depressive or grandiose, depending upon the previous personality traits.

Reaction Patterns.

(1) *Manic*: Patient presents largely the same symptoms as the manic phase of the affective psychosis except that all the symptoms are extreme and the delusions are more absurd. The individual shows flight of ideas, markedly increased restlessness, together with the presence of more or less grandiose ideas. He claims to possess innumerable millions, states that he is the strongest man in the world, possesses 500 houses, a thousand wives and numerous motor cars. As a rule the mood is markedly euphoric, although it varies very rapidly. This type is probably the easiest to handle as well as presenting the best prognosis under treatment.

(2) *Depressive Type*: This type resembles more or less the depressed phase of manic depressive condition. All the ideas are depressive in character. Patient appears to be very sad and his delusions are of sin and frequently

of negation, denying that he has any heart or brain or lungs. Here, too, the extravagance of his depressive delusions is the noticeable thing. All his movements are retarded, and it is difficult to get him to take sufficient nourishment. As a result the physical condition rapidly deteriorates.

(3) *Agitated*: In this type the motor excitement is so extreme that the condition is frequently described as galloping paresis. The condition would almost resemble that of an acute delirium with hallucinations and illusions and rapidly supervening exhaustion. Cases have been known to run a fatal course in two weeks.

(4) *Simple Dementing Type*: Here delusions are not very marked. Patients tend to show simply a progressive mental deterioration. They show no interest in anything, become more awkward and weaker and simply present a let-down of all mental and physical activities.

(5) *Paranoid Type*: This is not a very common type of mental reaction in General Paresis. The symptoms are essentially those of the Paranoidal State. Patient thinks that he is being ill-treated by everybody and is extremely irritable and disagreeable. Unless the neurological symptoms and the serology are carefully checked up the case may be considered to be one of paranoidal condition purely.

(6) *Neurasthenic*: In this type the patient is inclined to be more or less hypochondriacal, complaining of various aches and pains and demanding treatment for them. The condition very closely approximates the depressive type and might be quite reasonably included under that heading.

(7) *Schizophrenic Type*: In this type patient presents very often catatonic symptoms, such as mannerisms, negativism, stuporous and excited periods. Except for the neurological and serological signs patient would probably be put down as a case of Catatonic Dementia Praecox.

Tabo-Paresis.

This is a type of the disease in which the parenchyma of both the brain and spinal cord are involved. It occasionally begins as a tabes dorsalis and after a number of years the typical symptoms of general paresis are added indicating the further extension of the pathological process. In tabo-paresis the knee-jerks and ankle-jerks are absent, there is rhombergism, often a Babinski sign and an Argyll-Robertson pupil is present. To these are added the mental symptoms of General Paresis.

Juvenile Paresis.

This is a rather rare condition in which the disease appears quite early in life, in the teens or early twenties. Examination of the parents of such patients shows that either one or both are paretics or syphilitics. The mental picture presented is either that of mental deficiency or schizophrenia. The prognosis is extremely bad.

Treatment.

Up until within the last decade the treatment of General Paresis has been considered to be hopeless. In the last few years, however, as a result of certain new methods this view-point has somewhat changed. Occasional remissions have been observed ever since the disease has been known. These, however, were not very common. As a result of a survey of a thousand cases made in New York State Hospitals, it was found that spontaneous remissions occurred in only about 3.5%, and these remissions were as a rule of very short duration. As a result of modern methods of treatment the remissions have (to consider the results in a very conservative way) been increased to anywhere from 35 to 50% of cases depending largely upon the stage at which treatment is begun and upon the methods used by the clinician.

Various anti-syphilitic drugs have been used in the past and various methods of application have been in

vogue. I might mention the following methods which have been used with more or less success up until the last five years.

(1) Intensive intravenous treatment with arsphenamine or neoarsphenamine, combined with mercury intramuscularly and pot. iodide by mouth.

(2) The same treatment as (1) combined with frequent spinal drainage.

(3) The Swift-Ellis treatment or the sub-arachnoid administration or salvarsanized serum.

(4) Administration of salvarsanized serum into the cisterna magna or directly into the lateral ventricles of the brain.

(5) Administration of minute quantities of arsphenamine or neoarsphenamine directly into the subarachnoid space.

A certain measure of success followed upon all these methods if they were applied with some perseverance. The percentage of remissions was probably raised to from 10 to 25% at any rate by these methods.

Finally, however, the treatment of General Paresis has now pretty well narrowed down to two methods: (1) The *Tryparsamide treatment*, and (2) the *Malarial treatment*, or non-specific protein treatment.

(1) *Tryparsamide Treatment*: Tryparsamide is an arsenical preparation containing around 25% of arsenic and has been in use for about five or six years. It is a white granular powder, very easily soluble in water in clear solution. It is relatively non toxic and therefore can be given in as large doses as 3 grams. It is followed by practically no reactions and can be given quite safely in office practice. Tryparsamide is not an active spirochetocidal drug. It acts rather by stimulating the vital processes of the body and apparently raising the natural resistance of the body to the action of the spirochete. It also has the power of penetrating to a very considerable degree into the central nervous system. The only drawback to its use is the occasional effect which it has upon the optic nerve resulting in optic neuritis or optic atrophy. Before its use is decided upon the eyes should be carefully examined by an ophthalmologist.

The treatment is quite simple. The drug is dissolved in doses of from 1 to 3 grams in 10 to 20 c.c. sterile distilled water and injected intravenously once a week. From 12 to 20 injections constitute a course and then it is probably wise to give the patient a rest for a couple of months, during which period Bismuth hydroxide or mercury salicylate may be given intramuscularly. My own practice is to give the tryparsamide and the bismuth at the same time and then given a complete rest for six weeks to two months. Patients as a rule complain of no discomfort and very frequently gain materially in weight.

The results to be expected from the use of this drug are very good. Good remissions can be expected in from 35 to 50% of cases. I have patients under observation at the present time who have now remained well and able to carry on their regular occupation for four or five years. The danger of giving the drug is practically nil. I feel that every case of General Paresis should be given the benefit of this treatment, no matter how early or late it may be.

(2) *Malarial Treatment*: This treatment, which consists of the administration of malarial blood to the patient, was instituted in 1917 by Wagner-Jauregg in Vienna. Previous to the use of this method he had used various other nonspecific proteins such as tuberculin, sodium nucleinate, typhoid vaccine, etc. Tertian malaria is the type used for the treatment and this must be of a pure strain. Certain bad results have followed admixture with the quartan type.

The method of treatment is as follows: About 2 c.c. of blood is withdrawn from a patient suffering from tertian malaria who has not received

quinine and is injected either intravenously or subcutaneously into the paretic patient. In from 3 to 8 days in the intravenous method or from 1 to 2 weeks in the subcutaneous method the paretic patient develops inoculation malaria, characterized by marked chills and high fever. Depending upon the condition of the patient he is allowed to have anywhere from 9 to 16 of these chills and the plasmodium malariae is then killed by the administration of quinine, 10 grains being given three times a day for the first week and then once a day for the next week. Very often the malarial course is followed up by a course of tryparsamide.

The results of this treatment are also very good. Remissions running anywhere from 40 to 55 per cent. are claimed by those using the method. This method is used almost exclusively in the Vienna clinics, and the value of its contribution to medicine is evidenced by the fact that its originator was honored by the Nobel prize recently. The only drawback to malarial treatment is that it is much more dangerous than the tryparsamide treatment. The mortality rate is about 10 per cent. Cases have to be very carefully chosen, marked heart lesions, serious kidney lesions, pulmonary tuberculosis, extremely high blood pressure and extreme obesity, constituting as a rule contraindications to the treatment.

Finally, there is no doubt that in the tryparsamide and malarial methods, we have means at our disposal by virtue of which we can very materially alter the outlook of general paresis.

Notes on Cardiac Physiology Applied

(By J. A. Oille, M.B.)

Reserve

Cardiac Reserve is the amount of work the heart is capable of doing over and above the amount required to carry on the circulation at rest. Since the measurement of the actual output per minute during rest and violent exertion presents obvious difficulties, a consideration of oxygen consumption during rest presents obvious difficulties, a consideration of oxygen consumption during rest and muscular activity gives us some relative idea of the work the heart does, because this organ has to pump the blood that carries the oxygen. Oxygen utilization is something that can be measured with greater relative accuracy than actual blood volume output. In a lecture delivered last May, Douglas states that when a person is doing hard work the oxygen supply has to be increased thirty times over the resting requirement. This of course does not mean that the cardiac output has to be increased thirty times because a much smaller per cent. of the available oxygen is consumed during rest; only 240 c.c. oxygen per minute are absorbed during rest, while 4000 c.c. or more may be absorbed during violent exercise. One litre of blood carries 200 c.c., if fully saturated. Therefore, 20 litres of blood pumped per minute would supply 4000 c.c. oxygen; but all of the oxygen cannot be abstracted from the blood as it passes rapidly through the capillaries. If two-thirds be absorbed, then 30 litres a minute would have to be pumped. This gives one some idea of the tremendous reserve of the heart muscle. Some hearts can increase their output seven to ten times.

Now there are some important clinical deductions from this fact. (Axioms)

1. The reserve is so great that no increase of work due to valvular disease or high blood pressure can cause a

normal heart muscle to fail. Therefore, all cardiac failure is myocardial failure due to lessening of the reserve due to a damaged muscle, that is, a muscle weakened in some way, either by actual disease or interference with its nutrition by poor arteries or poor blood.

2. One can go further. The reserve is so great that seldom is the overwork caused by valvular disease or high blood pressure sufficient to cause much hypertrophy. Most of the hypertrophy one finds, therefore, must be due to a weakened muscle, for which the work of maintaining a normal circulation may be overwork. Thus overwork for the myocardium can occur in two ways: (1) from lessened strength, (2) from increased work to be done. Clinical evidence goes to show that the first is the more important. It is practically impossible to cause hypertrophy experimentally in a normal heart in animals. Hence we believe that muscle hypertrophies as it becomes weakened by disease or insufficient blood supply. Therefore, one concludes that the larger the heart, the more serious is the disease. The so-called "athletic heart" has been damaged by other factors than overexertion. Strenuous exertion, high blood pressure, or marked valvular defects cause very little hypertrophy in an undamaged heart muscle.

3. A third conclusion becomes at once evident. In all heart disease, excepting bacterial endocarditis, the prognosis depends on the condition of the heart muscle.

Then Douglas points out that just as the products of metabolic activity set the pace of the respiratory centre and thereby adjust the oxygen supply to suit the need, so the oxygen demands of the various organs must in some way regulate the circulation. It is obvious that the blood flow through the muscle, and especially the heart muscle, must be proportional to the metabolism. Either too little or too much blood would be harmful. One wonders whether the same laws govern the capillaries in the heart muscle as in skeletal muscles. Why do the capillaries open up so remarkably in working muscles?

1. Dale, Richards and Burn's theory of capillary dilation by some such substance as histamine, which produces dilation by antagonizing the constrictor effect of adrenaline or pituitrin. The latter idea is more or less an established fact, as Krogh has shown that capillary tone cannot be maintained if pituitrin be absent from the blood.

2. Next is discussed the relation of capillary tone in muscle to CO_2 and O pressure.

Haldane and Douglas think or thought that they had sufficient evidence to justify the view that the local circulation in muscles must be dependent on CO_2 and O pressure existing at the time, really the H-ion concentration in relation with the O supply.

Going with the capillary dilatation must be dilatation of small arteries to allow sufficient increase of blood flow. The exact mechanism by which this is brought about is uncertain. Krogh holds that there is a dilatation extending back from the capillaries to the arterioles by means of nerve control, that is an axon reflex. There is other evidence, however, that the small arterioles respond to chemical stimuli in a similar way that capillaries do, thus behaving unlike the large arteries. It would seem advantageous to have both capillaries and arterioles respond to the same chemical metabolic stimulus to increase blood by increased calibre, as dilatation of the capillaries alone would be insufficient.

Another important vascular reaction occurs when one set of tissues, as for instance the skeletal muscles, need more blood. If more blood goes to the muscles, there must be a compensatory constriction of the blood vessels in other parts of the body. This probably goes on in the intestinal tract, the liver, the spleen, and even the kidneys.

This must be brought about through vaso-constrictor nerves.

There are two factors involved in initiating such vaso-construction:

1. Lovèn Reflex.—Stimulation of sensory nerves causes vasodilatation in the area supplied, and vasoconstriction in other areas, that is, the rest of the body.

2. Increased CO_2 content (increase of H-ion concentration), or fall of O pressure in blood reaching the brain causes vasoconstriction in certain areas.

The above is a synopsis of vascular control regulating blood supply. We know there is vascular control, but the details of its operation are more a matter of theory than established fact.

It is both safe and easy to draw deductions from such a well known fact that the heart has an enormous reserve. But when it comes to making clinical deductions from theories, it is an altogether different matter. This constitutes the basis for many mistakes in medical teaching and practice. Where facts are lacking, theories must be advanced, investigated, and finally discarded or proven. This is one of the works of the laboratory. When the cause of a disease is unknown, its treatment must be based either on traditional experience with various remedies which is very great, or on a theoretical cause as new theories are advanced.

Let us discuss for a moment the situation in connection with high blood pressure. Here is an abnormality of vascular control of considerable importance. We know several diseases that elevate blood pressure, but there is a large group of people with persistent elevation of blood pressure, of which we do not know the cause. These cases of hypertension have been the subject of much theorizing that began before the blood pressure apparatus was invented. Each theory suggests a remedy, and the unfortunate part is that remedies continue to be used and to be believed in by the great mass of the profession years after the underlying theory was disproven. To mention an instance, consider the NaCl theory. A few years ago a physician published an article stating that in hypertension the blood pressure could be brought to normal by lowering the NaCl content of the diet below a certain figure. This was tried out by many investigators and disproven, yet the profession continues to tell high blood pressure cases to omit salt from their diet. Douglas failed to mention NaCl in his discussion of vascular control.

There is one lesson, however, in connection with vascular control, that should be pointed out. Even superficial consideration shows us that the part played by the vessels, especially the arterioles and capillaries, is equally important as that played by the heart, and many times more difficult to explain. We can at least appreciate that there is such an entity as vascular failure. This occurs in severe infections such as pneumonia, especially the pneumonias of the recent influenzal epidemic (1918-19). Here one would see a patient ill only a few days, lying comfortably in bed, not feeling ill, not short of breath, with a normal pulse, that is, a pulse of good volume and not rapid. The only striking thing about the patient was a peculior color, a mixture of pallor and cyanosis, greyish blue finger nails, etc. Then in a few hours, oedema of the lung would set in and the patient die of vascular rather than myocardial failure. This failure is also seen in shock.

It must play a part in the production of oedema in myocardial failure. One sees a case with a failing heart, especially in arteriosclerotic individuals who go along to a certain stage without oedema. Then all in a day a change occurs. The face appears pinched and thinner and oedema appears in dependent tissues and serous cavities in amounts such that it could not be dependent on fluid intake. In short, the vascular system including

the lymphatics, have lost control of the distribution of blood and body fluids and gravity assumed an abnormal influence.

Many other instances of other clinical examples of vascular failure could be cited.

The first theory of the cause of dilatation of capillaries in working muscles had to do with some chemical stimulation such as histamine. Physiologists have known for some time that various organ extracts would temporarily lower pressure. Two or three years ago some physicians endeavoured to apply this fact to clinical cases of hypertension in the form of a liver extract, assuming that if one kept the blood pressure normal the development of arteriosclerosis would be prevented, and cerebral haemorrhage averted. This assumption is probably not true, or only slightly true. It is probable that the increased strain of the vessel walls does act as an accessory cause of vascular disease.

To return to the liver extract, it is true no doubt that blood pressure can be temporarily reduced by its use intravenously, but the effects of the extract were so variable that its use has been largely discontinued. A few physicians, however, are still using it. One side of this story has not been written, namely, the possible danger of such treatment.

In discussing what opens up the capillaries in working muscles when the blood flow is increased so many times, one point that must be appreciated is that it is not increased blood pressure that forces them open. The blood pressure does not rise at all in proportion to the increased output.

The remainder of the lecture deals with the determination of the output of the heart. The output obviously is governed by the venous return. When the venous return is greatly increased by muscular exertion, by capillary and arteriolar dilatation, the heart increases its output by an increase in the number of beats and volume per beat per minute. There is some physiological controversy over the last point. Krogh and Lindhard, by their nitrous oxide method, estimated the cardiac output at rest at 3 to 5 litres per minute, and at 17.6 litres per minute when oxygen consumption was 1570 c.c.m per minute, and 35 litres per minute when oxygen consumption rose to 3200 c.c.m. While these figures may not be absolutely correct, they are relatively so, and show that the heart can increase its output 9 or 10 times.

The Haldane and Douglas method gave the output at rest at 7 litres per minute.

Yandel Henderson, in a third method, estimated the resting output still higher, e.g. 7.6 to 14.3 litres per minute, and with an O consumption of 4600 c.c.m. the output was 30.3 litres per minute. These variations show the difficulty of the problem. Evidently physiologists lack as yet an accurate method of estimating cardiac output per minute. When one is found, and if it be simple enough, it may have considerable clinical value.

At present the best guide we have to the output per minute is the character of the pulse. This is much better than the blood pressure apparatus. It is of course true that small pulse pressure is evidence of small or slow output, but vascular adaptation to output is so efficient that the output has to be very greatly lowered before it shows in the small pulse pressure. It can be detected much earlier by the finger. The output in cases of mitral stenosis and aortic stenosis must be greatly lowered, possibly down to 33 or 25 per cent. of normal. Life can certainly be comfortable and work done in the presence of a greatly lowered output. The blood supply of 3,000 c.c. at rest is much more than is necesasry. Metabolic requirements only remove 18 or 20 per cent. of the oxygen whereas 64 to 65 per cent, can be removed if needed. Therefore, the minimum figure of 3,000 to 4,000 c.c. per minute is just about three times as much as required. A feature of the above two valvular diseases is that on account

of the obstruction, the output per minute cannot be rapidly or greatly increased; hence, violent or prolonged exercise should never be undertaken. When the output is lessened, the whole arterial system contracts down so that the blood pressure is maintained unchanged, or may even rise above the normal, especially the diastolic. A small output per beat into a contracted arterial field raises the blood pressure in figures as much as normal. However, one detects the contracted arterial field and small wave of rise of pressure by the finger when the blood pressure figures read 120/70, or any normal proposition.

This is just an example of the fact that in medical training and practice physical examination is the one most important thing. It is true that in certain cases history alone establishes the diagnosis, and in others some laboratory procedure does, but in the great majority of instances physical examination is the determining method and thus the most important thing for a student to acquire is experimental physical examination and interpretation of the findings. This is founded on the basal sciences, physics, anatomy, physiology, and pathology.

The above lecture is based on a lecture by Professor Douglas of Oxford on the adaptation of the circulation to muscular exertion.

Pathology and Treatment of Cervical Catarrh*

(By M. B. Coutts, B.A., 278).

ETIOLOGY.

Catarrhal cervix is undoubtedly the most common ailment to which female flesh is heir. According to Strachan of Cardiff it is present in 75-80 per cent of parous women, and also in 25 per cent. of nulliparae. Other investigators give similar figures. Hence we must seek for the most frequent cause of cervical catarrh in the results of child-bearing.

A certain number of cases of cervical catarrh are congenital. The mucosa of the portio in its development is identical with the musosa of the canal, the squamous epithelium growing over the portio in the latter months of intrauterine life. In some cases the growth of squamous epithelium is not complete, and the columnar epithelium covering the cervix is left exposed to the acid secretions of the vagina, the irritation subsequent upon walking and other movements, and becomes infected. Congenital catarrhal cervices belong to the "papillary type" which will be described below.

Bacteriology.—The immediate cause of cervical catarrh is almost invariably infection. The vagina normally is the home of a rich bacterial flora, but these bacteria are saprophytic and not pathogenic. This is due to the fact that there are no glands in the vagina which can harbor pathogenic bacteria, and also because of the normal acid reaction of the vagina which inhibits the growth of most pathogens. This acid reaction is maintained by the activities of Doderlein's Bacillus, and by the fermentation of the glycogen in which the tissues of the vagina are peculiarly rich. This acid reaction is most apt to be interfered with just after labor when the lochia renders the reaction alkaline.

The organisms most commonly found in catarrhal cervices are—Streptococcus, Gonococcus, Staphylococcus, and B. Coli. The first two named are doubtless the causative agent in the vast majority of cases, but authorities differ as to which should occupy the place of honor as the most frequent cause.

*Extract from Gynaecological Thesis, "Cervical Catarrh," 1928.

Trauma.—By far the most important factor in the determination of infection to the cervix are the traumata incident to childbirth. There is no birth without some laceration of the cervix, either gross or microscopic, as is evidenced by the inevitably changed appearance of the parous cervix. The lacerated cervix is edematous, a certain number of bacteria are invariably introduced during labor, and the alkaline lochia furnishes a suitable medium in which they may thrive and multiply. Any operation which involves mutilation of the cervix also furnishes a point of entry for bacteria. The exposed surface of the cervix becomes the seat of infection. The wound gapes on account of the unopposed action of the longitudinal muscle, after the circularly disposed fibres have been torn (Ectropion). Then follows the sequence of events described below.

Gonorrhoea.—Catarrhal cervix may arise without trauma, either physical or chemical from the activities of the gonococcus. This organism is not inhibited by the alkaline cervical secretion, and demonstrates its fondness for epithelium by its ability to penetrate the uninjured surface of the cervical canal and set up a chronic infection. The organism lies between the cells in the glands and defies all efforts to dislodge it. The cervix is the third most common seat of gonorrhea in the female.

Other Etiological Factors.—Chronic ill health and debilitating diseases commonly lead to the establishment of inflammatory foci in the cervical glands with the resulting pathological changes. Lack of hygiene in the care of the genitals or too strenuous hygiene from the too frequent use of strong antiseptics also occasionally account for infection. Trauma due to the use of pessaries and certain contraceptives play a part. Masturbation may result in introducing infection from the external genitals. —(Burns.)

PATHOLOGY.

Gross Appearance.—Clinically the catarrhal cervix presents a picture the most prominent feature of which is the reddening of the portio round about the external os. This surface is often granular, bleeds very easily, and shades off into the surrounding normal cervical epithelium. The red color is redder than the color of the normal cervical canal, and stands in sharp contrast to the pale pink color of the healthy portions of the portio. This appearance is known as "erosion," and is of varying extent, sometimes covering the whole of the portio vaginalis. A thick abundant secretion of viscid glairy mucus, sometimes purulent is always present. Hypertrophy of the cervix, and the presence of hard tense cysts (Nabothian Follicles) are frequent.

Acute Catarrhal Cervix.—Catarrhal cervix may be either acute or chronic. When seen it is usually chronic having passed through the acute stage. In the acute stage, the inflamed cervical mucous membrane becomes edematous and swollen. A great increase in secretion takes place. This may be purely mucoid or mucopurulent in character, and may be tinged with blood. Dense infiltration of the interglandular stroma with leukocytes is the main lesion in acute inflammation. As the subacute and chronic stage is reached an increasing number of plasma cells is encountered. The glands increase in size. Their outlets are often obstructed on account of the thick secretion, the inflammatory reaction choking their outlets, and retention cysts results. The lining epithelium becomes cuboidal, and the cysts may honey comb in any direction, increasing the portio to large dimensions and presenting grossly the appearance of a tumor. Necrosis and sloughing due to inflammation may produce stenosis of the canal.

The Stages of Chronic Catarrh.—Pathological Types.—According to Frank who has made a very extensive survey of the literature of obstetrical and gynecological pathology we have the progress of an erosion divided into three stages; viz.—Ulceration, early repair, complete repair.

In the early stages we have an infil-

trated denuded area on the portio due to the maceration and necrosis of the surface layer of squamous epithelium. The papillae under the epithelium are also densely infiltrated with round cells. Cervical glands grow downwards from within the cervical canal, and approach the denuded area from below. This is the stage of true erosion, and is rarely seen. Gilbert I. Strachan of Cardiff, and Toronto workers however state that in all sections of catarrhal cervix examined by them there has been some ulceration in some portion of the section. It was thought at first that the red areas seen were always ulcerated, and hence arose the term "erosion."

In the early stages of healing the bright red area is covered by high columnar epithelium with oval basal nuclei. The cells stain like true cervical glands described above. Below the surface are more or less numerous branched glands. The picture is that of a portion of the cervical canal transported bodily to the portion plus inflammation. If the surface is smooth, the glands few in number and not dilated, the erosion is simple. If the glands are numerous and dilated, and lined by high columnar epithelium, often assuming papillary forms, and containing goblet cells, a follicular erosion is said to exist, if the gland elements are numerous, run downward, parallel to each other, a papillary appearance is presented, because of the numerous stroma papillae which project upward between the glands. Congenital erosions are of this type.

The third stage is characterized by an increase in the squamous epithelium which grows over the mouths of the glands, plugging them, and causing retention cysts. The epithelium of the glands become cuboidal and they enlarge greatly. The final healing takes place when the cervical glands have been converted into solid epithelial plugs arising from down-growths of the squamous epithelium, which eventually flatten out into the general surface covering. There is however, little tendency to this type of spontaneous cure. The increase in the size and number of the glands may produce localized or diffuse tonsil like hypertrophies, with irregular tonsil-like surfaces, which may reach large size and resemble adenoma.

The extremely varied configuration presented by the intermingling of the two types of epithelium on the surface and in the glands produces a picture of epithelial proliferation closely resembling carcinoma. At times the diagnosis is doubtful.

Complications and Sequelae.—The complications and sequelae of this condition are important, since they are responsible for many of the symptoms, and are in themselves sufficient reason for demanding vigorous treatment of the lesion as early as possible.

1. Pelvic cellulitis—the infective process if left to work its way unchecked may spread via the lymphatics into the tissues of the broad ligaments. This will give rise to pelvic discomfort and premenstrual pain. The ovary and the peritoneum might conceivably be infected, with characteristic symptoms.

2. Involvement of the utero-sacral ligaments, giving rise to dyspareunia and backache.

3. Anemia—possibly due to chronic toxemia.

4. Arthritis—has been stressed of late by Laura Moench of the Mayo Clinic. Thirty-seven per cent. of cases of chronic endocervicitis harbor anaerobic strep. in virulent strains, which are identical with strains cultured from cases of arthritis in the same individuals.

5. Salpingitis—particularly in cases of gonococcal cervicitis. The cervix is a lurking place for these organisms which will infect the tubes just after a menstrual period or after labor.

6. Carcinoma—it is not proven that carcinoma is a sequelae of cervical catarrh, but when we consider that there is a high incidence of carcinoma in parous women as compared with nulliparae, and the frequent difficulty pathologists have in differentiating the

epithelial melange presented in erosion of some types from carcinoma, we may be justified in considering cervical catarrh as a predisposing factor in carcinoma of the cervix.

Clinical Symptoms.—These are: Leukorrhoea, sterility, general debility, lassitude and constipation, backache, pain in the left side, menorrhagia, premenstrual pain, and dyspareunia.

Clinical Signs.—The cervix may appear quite normal except for the catarrh (Burns). More often, however, one of the types of erosion described under the pathology of the condition is present.

Clinical Types.—We may divide these cases clinically into three types.

1. Those due to laceration followed by irritation and adenomatous overgrowth of the cervical epithelium.

2. Those primarly due to gonorrhoea.

3. The so-called primary erosion of virgins—usually the erosion is of the papillary type and gonococci cannot be found. There is no laceration. General ill-health or masturbation are often predisposing foctors.

TREATMENT.

Since Emmet began his observations in 1862 the treatment of chronic endocervicitis has been the bugbear of all conscientious gynecologists. The condition is extremely invidious to the patient, and has proven well-nigh invincible to the physician. The multiplicity of treatments suggested indicates that none of them are entirely satisfactory.

Treatment of this condition may be considered under the following heads: Palliative, non-operative, operative.

Palliative Treatment.—This method includes the application of chemical caustics and antiseptics to the inflamed region. Treatment must in all cases be prolonged, and results are uncertain. However, it is the only method at the disposal of many physicians, and the best technique for this procedure should be familiar.

The cervix should be exposed in a good light, and the discharge wiped away. An attempt should be made to destroy all the diseased tissue possible with pure carbolic acid, alcohol being used to stop the action of the carbolic. Then apply some caustic such as silver nitrate, 10 per cent. Churchill's iodine, nitric acid, formalin or copper sulphate. The caustic should be applied right in the cervical canal. Any Nabothian follicles present should be punctured. A daily douche should be taken by the patient.

The treatment should be repeated weekly for from eight to ten weeks. There is less than a 50 per cent. chance of cure, and the patient should be warned of this.

Burns considers that the only proper way to apply antiseptics to the cervical canal is by the use of ionization. He uses a zinc anode which he inserts into the cervix, and surrounds with a solution of zinc sulphate, which is soaked in a tampon of gauze. The gauze is left in the vagina for four hours after the operation. He uses a current of twenty milliamps for from 10-15 minutes. He applies this treatment three times a week. He found that this procedure was of benefit to patients with cervical catarrh without erosion (65 per cent. of cures after three months), but obtained only 12 per cent of three month cures in cases of patients with erosion.

Non-operative Treatment.—Several methods of non-operative treatment have become very much the vogue of recent years. The use of the cautery in particular has become very widespread. The Paquelin cautery was used to a wide extent, but its use has dropped from fashion of late years. When the actual cautery was introduced several methods were employed. Searing over the erosion has been found to be unsatisfactory. A better method was to heat the cautery to a cherry red, and then plunging it into the cervical canal, leave it there for some time. By this treatment the diseased tissue was destroyed, and a hyperemia of the cervix was produced which combatted the in-

fection. However, the extent of the burn could not easily be controlled.

The most recent refinement in cautery treatment is the use of the nasal type cautery tip, and since such encouraging reports of its use are being received, it will be described in some detail.

This treatment can be performed in the office, and without anesthetic. If it is found that the patient is nervous, nitrous oxide analgesia, self-administered can be employed. The cautery is inserted into the canal to a varying depth according to the extent of the pathological process, and drawn outwards through the tissues in a direction radial to the cervical canal. Six of these cuts are made through the diseased tissue in lines about 3-16 inches apart, and situated at 2, 4, 6, 8, 10, 12 o'clock. Two or three treatments at monthly intervals are sometimes necessary to effect cure.

After cautery treatment there will be an increase in discharge for two or three weeks. A pad will have to be worn and the next menstruation will be profuse. A pack may be necessary. The vagina and cervix should be cleaned out every ten days and the cut area painted with alcoholic mercurochrome. Failure to do this may result in stricture. Sex trauma should be avoided for two weeks. The vagina should NOT be douched.

Davis of Milwaukee claims that by this procedure all of his patients are improved, and 80 per cent. of them are cured. This is surely remarkable if other men can repeat his results. The rationale of the treatment is in the destruction of a large part of the diseased tissue, in the production of hyperemia of cervix, and in that the scar tissue formed chokes off the blood supply to the rest of the diseased tissue, which was not directly exposed to the cautery.

This treatment is suitable only for chronic cases. Experience has shown that acute gonorrhoeas, and cases giving a history of salpingitis flare up under cautery treatment. Hence cautery is contra-indicated in these cases.

Blair of Vancouver combines the treatment above outlined with a treatment introduced by Nassauer in 1907. After treating with cautery he introduces a hygroscopic powder into the cervical canal and vagina by an instrument known as a "Siccator." The powder consists of kaolin two parts, to sodium bicarbonate one part. The cauterization does not have to be repeated, but he introduces the powder for four treatments, each two weeks apart.

Radium treatment has also been successfully used in some clinics. It is particularly valuable in patients past the menopause, and particularly when the differential diagnosis between erosion and cancer is in doubt. One hesitates to use it during the child bearing period on account of the danger of bringing about an artificial menopause, and if one uses a safe dosage there is a danger that the desired result will not be obtained.

Vaccines are variously described as all the way from "utterly useless" to "an extremely valuable adjuvant to treatment." Those who recommend vaccines say that they should preferably be autogenous. They are said to be most valuable in cases complicated by the presence of the gonococcus. However, the most that is ever claimed for them is as an "adjuvant" to other treatment.

Diathermy—the use of one pole of the diathermy instrument in the cervix, and the other in the form of a metal belt around the body with the maintenance of a temperature within the cervical canal of 114 degrees Fahrenheit for ten minutes is claimed to destroy all gonococci present. However, it does not destroy the other organisms which are invariably present, and has no effect whatever on the adenomatous overgrowth of cervical glands. However, Justina Wilson of St. Bart's, London, claims that diathermy, used according to her method yields the most rapid, sure, and efficient results in the treatment of gonorrhoea of the cervix. If erosion be present she uses the

water cooled ultra violet light on the croded area. It appears to the writer that the use of diathermy would be theoretically sound in cases of acute gonorrhoea.

Operative Treatment.—Many gynaecologists of great surgical skill, among whom is Matthews, maintain that all the procedures outlined above are merely palliative, and that the only logical method of treatment is operative with the removal and repair of the whole of the diseased area. Several operations have been described which will be reviewed briefly.

Emmet devised an operation known as the Emmet trachelorrhaphy, and which is still done by some operators. It is most suited to lacerated cervices where the eroded area is small. The sides of the laceration around which there is erosion are cut away, and the raw surfaces brought into apposition and stitched over. This operation is effective in only a small proportion of cases since the operation wound is often re-infected. It is useless when there is extensive hyperplasia of cervical epithelium and glands.

Schroeder devised an operation which is known as amputation of the cervix, or rather, high, amputation of the cervix. This procedure is radical since the whole of the cervix is removed, the cervical canal remaining very short. This operation is objected to strongly by those who consider that the cervix has an important function to perform in preventing the spread of infection to the endometrium, and in pregnancy. Blair claims that normal pregnancy is difficult to carry to its termination without a cervix, but that amputation is less objectionable after the menopause.

The operation now held in highest regard by gynecologists is the Sturmdorff coning operation. Sturmdorff bases his procedure on the premises that all the cervical canal is a potential seat of infection, the depth of the infection becoming progressively less as the internal os is approached. He also holds that the function of the cervix can be conserved if most of the muscle tissue is left, and that an operation which would remove the gland-bearing tissue and preserve the muscle intact would be ideal, and would allow of subsequent normal pregnancy. To this end he excises a cone of tissue, the base of which is at the portio, and the apex of which is near the internal os. This operation leaves most of the muscle tissue. The raw area is covered over by squamous epithelium through the medium of an ingenious method of stitching,—the Sturmdorff double inverting stitch. Matthews claims the following results from a series of 200 operations: Cured, 64 per cent.; improved, 28 per cent.; unimproved, 8 per cent. These results are as good as those obtained by Kelly at the Johns Hopkins Hospital where the Schroeder operation was used.

At the Toronto clinic operation is considered the best method of dealing with catarrhal cervix, and it doubtless produces the surest and most prolonged results. The disadvantage of operative procedure is that it requires hospital surroundings, anesthetist, etc., and is very expensive. We must consider the tremendous number of women afflicted, and continue to seek for some effective means of treatment which may be made readily available for all.

Summary.

1. Cervical catarrh is a clinical condition characterized by leukorrhoea, occuring in almost all cases of endocervicitis, and frequently accompanied by erosion of the cervix. It is extremely common, almost universal.

2. The cause of cervical catarrh is infection, and the predominating feature in the pathological picture is an adenomatous overgrowth of the epithelium of the cervical canal.

3. Leukorrhoea is the most prominent and distressing symptom, and carcinoma is the most dangerous sequela.

4. Treatment is extremely difficult. Operation gives the best results, but the use of the nasal type cautery appears to be the method of choice for most physicians.

Treatment of Syphilis

By DR. E. J. TROW, M.B., Assoc. Prof. of Medicine, in Charge of Dermatology.

This chronic infection, provoking as it does a variety of lesions, presents great difficulties in successfully bringing about its complete elimination. In addition to the therapeutic difficulty the probable wide difference in virulence of the invading organism and the resulting type and severity of the tissue reaction must be considered as bearing on the ultimate result. The question of immunity is still a much discussed point and if one believes with Neisser and his associates, following prolonged investigation of syphilis among apes in Java where he concluded that a resistance to a syphilitic infection depended upon the persistence of the original infection and said "there is no immunity in syphilis without infection, and if there is infection there is immunity." Following this thought further Neisser came to the conclusion that the behaviour of a syphilitic animal that had been treated might, by a second inoculation with syphilis, be utilized to determine the value of the treatment employed; that is to say that, if after treatment the animal could be reinfected, he considered the first infection radically cured. For a long time these views were accepted, but following a long series of experiments with rabbits Chesney and Kemp suggest that in rabbits at least there may develop, during the course of a syphilitic infection, an immunity which persists after the infection has been eliminated and that acquired immunity in syphilis need not depend on active foci as suggested by Neisser. Many investigators have stated that treatment itself affects the immune state of the infected person. The frequency of neuro-recurrences in patients badly treated with infrequent and inadequate dosage may be explained on this basis, and it seems reasonable that it be so. Brown and Pearce conducted some experiments with rabbits, and their conclusion regarding animals treated with subcurative doses of the arsenicals was that the course of the disease in animals so treated was more severe after treatment than in the control animals. They thought, too, that not only the treatment itself but the time at which treatment is started during the course of the infection had a bearing on the immune process.

Many times in animal experiments it has been shown that rabbits treated before the forty-fifth day were usually susceptible to a second inoculation, whereas those treated after the ninetieth day could not be reinoculated. These facts may mean that those animals treated early were cured and the ones treated late were not cured; or it may mean something more obscure, and it has been suggested that early treatment prevents the development of the immune state.

In the special treatment clinic of the Toronto General Hospital it has been noted that a reinfection is not a particularly rare occurance where the first infection was treated in the sero-negative, dark field positive stage.

The whole question of immunity in syphilis, therefore, is quite confused and it is likely that many things such as age, sex, general health, treatment, tissue reaction and many other factors are all concerned in its production.

In discussing treatment it is not possible in a short paper to go into the various plans of treatment adopted, but a few observations may be of interest.

In treating early syphilis, which usually occurs in fairly young and often healthy adults, it is important to remember the infectiousness of the disease and therefore, I feel that such cases should be detained in hospital for a considerable period, as in doing so energetic regular treatment can be carried out; the patient can be more easily convinced of the seriousness of the infection and the public may be

protected from this infection. After treatment, for short periods, relapses of infectiousness may appear, for short periods, and it is well to remember that this may be so.

Those who treat syphilis realize that no combination of drugs now available can be guaranteed and it is just as well not to speak too freely of cures, but rather try to impress the patient with the necessity of a prolonged period of treatment and observation.

A group of patients suffering from primary and secondary syphilis differ greatly from a group affected by a long standing infection; and therefore, it is always advisable to give individual consideration to each case.

Usually the early syphilitics are free from extensive involvement of the important viscera and from degenerative types of lesions due to advancing years.

It is essential that a diagnosis be established as early as possible in the course of the disease and when established treatment proceeded with immediately, and for this treatment a wide range of drugs is available.

It has been generally accepted that in the treatment of early syphilis one of the arsenicals, Mercury and Potassium Iodide be employed. If an arsenical and mercury are used simultaneously it is obvious that the patient must have rest periods as there is a limit to a patient's tolerance and it would seem then that a course of an arsenical followed by a course of mercury and this in turn followed by a second course of an arsenical and mercury may be more valuable than the combined form of treatment with rest periods. It is true that many early cases are probably cured by one course of treatment, but the majority of our cases require more prolonged treatment. When treatment is begun in the early secondary stage the progress of the lesions and the general tissue reaction is suddenly interrupted and the development of the patient's own resistance against the spirochaetes is prevented; therefore, in the case of a combined type of treatment the patient enters upon his first rest period without adequate resistance against any living spirochaetes remaining in his tissues. Depending upon where these living organisms are and the length of the rest periods various clinical recurrences may be seen or he may show signs of a neuro-recurrence.

In a recent survey of sixty-one cases of treated early secondary syphilis in the special treatment clinic of the Toronto General Hospital nineteen cases of some type of neuro-recurrence appeared as well as several clinical recurrences in other tissues. Most of these recurrences could be readily explained by inadequate dosage, or prolonged rest periods; but in a few particularly virulent infection seemed to be the cause. It would seem reasonable then that our treatment of early syphilis be more or less continuous, i.e., one drug followed by another with no prolonged periods of rest; at least not for the first year. I am convinced that most cases of early syphilis are undertreated and given too long rest periods.

In long standing infections or late syphilis where one is dealing with patients older in years—many of them suffering from disabling lesions of the viscera, nervous or cardiovascular systems it is essential that great care be used in treatment. It is sometimes more important to build up such a patient by rest and tonics in order to maintain his general health than proceed immediately with any particular antisyphilitic remedy, important as that may be. The variety of clinical symptoms due to a long standing infection may necessitate a wide range of types of treatment to be adopted, such as special forms of treatment for neuro-syphilis, etc.

In conclusion let me say that it would seem inexcusable to treat syphilis merely to control temporary infectious lesions, but rather one should approach the disease in its broadest sense having regard for the patient, his family and the public. Prolonged follow-up and accurate observation is as essential as the more specific forms of treatment.

Blood in the Urine

James C. McClelland, B.A., M.B.

Of the urinary symptoms, haematuria is extremely important. Once this appears one should make a thorough search for the cause, bearing in mind that early treatment to a bladder tumour can completely and easily destroy it. If this should be allowed to progress, it may not be found until it has become malignant, and treatment is much more difficult.

Symptoms.—Blood may appear in the urine without the patient having any other symptom. Geraghty in his series of bladder tumours showed that in seventy-six per cent. the first sign was a "symptomless haematuria." Frequency and pain very often accompany bleeding, and when present indicate infection some place in the urinary tract. Pain may be present in the flank, dull, sharp or so severe as to be called a renal colic. Pain in the kidney area accompanied by blood in the urine points to the kidney as the source of the trouble, unless there is an obstruction preventing free access of urine to the bladder,and the damning back of urine in the renal pelvis, producing a painful distension. For example, this occurs when an infiltrating tumour of the bladder surrounds the ureteral orifice producing a carcinomatous stricture. Renal and ureteral colic will localize the trouble, and one must bear in mind that a calculus passing down the ureter is not the only thing which will produce pain. A blood clot, ureteral stricture, a piece of tumour, or some caseous material will also produce a ureteral colic. Pain in the suprepubic area accompanying haematuria may point to the bladder as the origin of the bleeding; a distended bladder may mean retention, or the pain may indicate the contracted bladder of advanced vesical tuberculosis. Pain referred to the end of the penis usually means an inflammation of the trigone from a variety of causes, and is often accompanied by a terminal haematuria. Burning during urination usually means infection with involvement of the urethral mucosa. Therefore, it is important to note if any of these urinary symptoms are present, as they often aid in localizing the source of bleeding.

Urine Examination.—The urine collected into two glasses is often of great help in determining the source of the bleeding. One may have blood in the first glass and a clear second, indicating that the blood was coming from the urethra. Blood intimately mixed in both glasses points to the bleeding occurring in the bladder or the structures above, namely, the ureter or kidney. A clear first glass and blood in the second points to the origin of the bleeding being in the bladder or posterior urethra.

The causes of haematuria may be classed under three main headings:

1. Causes of bleeding *in* the urinary tract.
2. Causes of bleeding *outside* the urinary tract.
3. *Systemic* causes of bleeding.

(1) *Causes of bleeding in the urinary tract.*—These may be considered under the headings of: Inflammation, tuberculosis, new growths, calculus, trauma, and applied to each of the structures making up the urinary tract, as kidney, ureter, bladder, prostrate and urethra. (See table).

Kidney.—The inflammations in the kidney are pyelonephritis and pyonephrosis, varying degrees of the same process. Microscopic blood occurs more frequently than gross bleeding. Renal tuberculosis has microscopic blood as a frequent finding. Occasionally there is a good deal of gross blood. When this occurs, the bladder is usually the seat of an advanced tuberculosis, secondary to the kidney lesion, and

	Kidney	*Ureter*	*Bladder*	*Prostate*	*Urethra*
Inflammations.	Microscopic but occasionally gross.		Acute: gross and microscopic. Chronic: Microscopic.		Gross and Microscopic.
Tuberculosis.	Microscopic (constant) Occasionally gross.		Microscopic and gross.		Microscopic.
New Growths.	Gross blood and a mass in the flank.	Gross.	Gross.	Gross and microscopic.	Gross.
Calculus.	Microscopic and gross.	Microscopic and gross during colic.	Microscopic and gross.	Microscopic Bloody ejaculations.	Gross and microscopic.
Trauma. Uncommon Causes.	Gross. Hydro-nephrosis. Polycystic disease.	Stricture (rarely).	Gross. Bilharzia diverticulum.		Gross.

literally "weeps" blood. An acute cystitis of this variety must not be looked upon as the lesion to be treated while the main one in the kidney is overlooked. Neoplasms produce blood in the majority of cases and show a mass in the flank, which may reach large proportions. Stones in the kidney may lie dormant for a long time without symptoms, but usually there is some pain varying from a slight dull ache to a very acute colic. During the pain and particularly the colic, either gross or microscopic blood is usually present in the urine provided the tear communicates with the pelvis. Polycystic disease may give haematuria. Hydronephrosis very uncommonly has bleeding.

Ureter.—A small calculus passing down the ureter usually causes bleeding, either gross or microscopic. The urine from a patient with a ureteral colic should be centrifuged and a careful search of the sediment made to determine the presence or absence of red blood cells. New growths from the kidney pelvis or bladder may extend into the ureter and be a source of bleeding. Stricture of the ureter has pain as its most common symptom, but occasionally there may be a small quantity of microscopic blood.

Bladder.—Acute and chronic cystitis have blood in the urine commonly. An acute cystitis may have a great deal of gross blood coming at the end of urination, or intimately mixed with the urine. The chronic type may have a small number of red blood cells in the urine, but pus predominates usually. Vesical tuberculosis gives blood almost constantly. An early lesion produces microscopic blood, and an advanced lesion, gross blood. The mucosa of a late tuberculous cystitis is fiery red in colour and literally "weeps" blood into the bladder cavity. A stone in the bladder causes an injury to the bladder wall, producing haematuria and pain at the end of urination referred to the tip of the penis, due to the bladder closing down and pressing the stone against the sensitive mucosa of the trigone. Injury, whether caused by an instrument, or an accident, produces a varying amount of bleeding, according to the extent of the damages. Individuals who have lived in the tropics, and particularly in

Egypt, may have their bleeding from bilharzia haematobia. This is small in amount, and diagnosed by finding the parasites in the urine, and the bladder gives the picture of small glistening yellow nodules and a few areas of granulation. A diverticulum is usually the site of a cystitis and bleeding may be caused by this inflammation.

Prostate.—Adenomyoma and carcinoma of the prostate produce blood, both small and large in amount. Bleeding is relatively more prevalent with the adenomyoma than the carcinoma. The bladder mucosa resists infiltration or carcinoma of the prostate, though ulceration and bleeding does occur in a few cases. Stones in the prostate proper do not produce a definite haematuria, but are a source of pain to the patient and evidenced by bloody ejaculations. Injury will give bleeding and vary according to the amount of injury.

Urethra. — Acute urethritis has bleeding as a symptom occasionally. Chordee may be so severe as to rupture an inflamed portion of the urethra and cause a good deal of bleeding. During treatment for acute gonorrhoea, strong chemicals used in a very sensitive urethra will cause considerable pain, frequency, tenesmus and haematuria. The trigone of the bladder is usually involved in this acute process. Tuberculosis of the urethra occurs along with tuberculosis in the other parts of the urinary tract, and may produce stricture in young male patients. New growths of the urethra are very uncommon. Tumours from the bladder occasionally grow down the urethra or a carcinoma of the penis invades the urethra to produce blood in the first glass urine. A calculus may be passed through the urethra, causing damage to the urethral mucosa, as it is forced along to the meatus. Trauma—violent injury to the urethra caused by falling astride some narrow object as an iron bar or a fractured pelvis, causes a rupture of the membranous urethra. Blood appears at the meatus as the first symptom. The passage of instruments through the urethra frequently causes bleeding even though the very greatest care is taken to avoid it.

Nephritis is a frequent cause of microscopic bleeding and occasionally gross bleeding casts and albumen are usually the companions of the blood, and serve to help in the diagnosis. Drugs, such as santal oil, turpentine, cantharides, carbolic, etc., produce red blood cells in the urine in varying quantity, gross and microscopic. Varicose veins may occur anywhere in the urinary tract, rupture and produce very acute bleeding. This is very uncommon. A naevus occurs rarely, but when it does one has considerable difficulty controlling the haemorrhage.

(2) *Causes of bleeding outside the urinary tract.*—Inflammation and new growths bordering on the bladder may involve it in that process. Acute pelvic inflammation as occurs from an inflamed appendix hanging over the brim of the pelvis with rupture, salpingitis and inflammations in the broad ligament, new growths in the bowel, cervix or body of the uterus, may involve the bladder and give the signs and symptoms of a local lesion there. Later, infection occurs or a fistula forms when the bowel contents are discharged through the bladder, or urine finds its way through the fistula into the vagina as in the case of carcinoma of the cervix. In the male, carcinoma of the rectum invades the bladder and produces a recto-vesical fistula.

(3) *Systemic causes of bleeding.*—Emboli from an endocarditis reach the kidneys, producing small infarcts, and cause sudden pain in the flank followed by blood in the urine. Leukaemias, purpura haemorrhagica, scurvy, haemophilia and focal infections may cause bleeding. Their diagnosis is made by a general physical examination taking particular note of the heart and spleen. Blood smears and blood cultures are also important. Similar to epistaxis in arteriorsclerosis, one may have blood in small or large amounts from the kid-

neys. Foci of infection occasionally feed organisms to the kidneys, and blood occurs in the urine.

Alkaline Urine.—This is a common cause of burning and frequency. At times the alkalinity may increase and produce a great deal of irritation, together with microscopic or gross blood in the urine. It can be corrected by acidifying the urine with large doses of acid sodium phosphate.

Diagnosis.—When a patient presents himself, complaining of "blood in the urine," how is one to proceed to reach a diagnosis? A good "history" must be elicited and an effort made to localize the trouble to the urinary tract, around the urinary tract or a general disease. This is not always possible, but the occurrence of frequency or pain will help considerably.

Physical Examination.—This is very important and should include every system in order. Particular care is to be taken to make a routine rectal examination. This should include an inspection of the anus for haemorrhoids, fissures, and fistulae. The tone of the sphincter is to be noted, particularly as a relaxed sprincter is present in spinal cord disease which impairs the emptying power of the bladder. The membranous urethra is felt between the finger and the under surface of the symphysis. Both sides of the membranous urethra are palpated by approximating the index finger inside, with the thumb outside, and any tenderness or induration noted. This manoeuvre helps in diagnosing an inflammation or abscess in Cowper's glands, or the region around it. By following up the membranous urethra, the tip of the prostate is reached. In the prostate, note the size, presence or absence of the median furrow, consistence of each lobe, any induration and where it extends to. At the upper border of the prostate the seminal vesicles, bladder wall, vasa deferentia and lower side of the ureter are found, but if normal they are difficult to distinguish. Induration in any one of them makes it prominent and easily recognized, for example, the infiltration of the base of the bladder by a tumor. The rectum mucosa as far as the finger will reach is carefully explored for masses and areas of induration.

The presence of abnormalities in the urine accompanying the blood, such as pus, organisms, casts, etc., are of great aid. Pus points to an inflammation commonly within the urinary tract and occasionally secondary to a lesion of a neighbouring organ. Pus with no organisms commonly points to the lesion being tuberculosis and the tubercle bacillus should be looked for. Blood with albumin and casts indicates a nephritis.

An X-ray of the kidneys, ureter and bladder (K.U.B.) is done to show the presence or absence of a stone. The size of the kidney may also be outlined.

When as much information as possible has been collected by the above methods, and a definite diagnosis has not been made, cystoscopy must be done for inspection of the bladder, and a study of both kidneys by means of the separate urine. Functional tests can be carried out with each separate kidney, and the pelvis injected with an opaque fluid to get an outline of the kidney pelvis and ureter (pyelograms). Dilations of the kidney pelvis, kinks of the ureter, and the exact location of stones can be noted. Tumours of the kidney invading the pelvis cause a filling defect in the pelvic outline. pyelograms are also a great aid in ascertaining whether an opaque mass in one area is connected with the kidney or not.

Essential Haematuria.—After a thorough search for the cause of blood in the urine has been made, and the source not found, we fall back on the term idiopathic or essential haematuria, meaning a confession that the cause has not been found.

In conclusion may I point out that blood in the urine tends to appear intermittently and once it occurs it must be considered serious until proven to be otherwise. Early diagnosis is necessary to allow proper steps to be taken

to remove the cause before it is beyond treatment. The bleeding tends to be intermittent and the periods between attacks allow the condition to advance so that an early diagnosis is very, very important. *Therefore, all patients complaining of blood in the urine must be thoroughly investigated during the first attack and no time lost waiting for further bleeding during which time the condition progresses.* The best example of the advantage of early diagnosis, is a benign papilloma of the bladder which is easily destroyed when recognized early, but later it becomes malignant and is much more difficult to cure.

Haemorrhagic Disease of the Newborn

(*By T. H. GLEESON, 2T8*).

DEFINITION.

A disposition to bleeding is seen with many diseases of the first few days of life, especially those of an infectious character like syphilis and pyaemia—with most of these, the haemorrhages are small and the condition may be compared to the haemorrhagic tendency seen in certain forms of infection of later life, such as measles, smallpox, and malignant endocarditis. There is, however, a class of cases in which the haemorrhages are not associated with any other known process, and *in which the escape of blood from the small blood vessels is the chief or essential symptom.* In these cases the haemorrhage is much more extensive than in the others mentioned. These haemorrhages are characterized by the fact that they are spontaneous in origin, having no connection with delivery, they are multiple in location, they tend to cease spontaneously after quite a limited time, but they are often greatly influenced by treatment. They are most often from the mucous membranes of the stomach and intestines, or from the umbilicus or beneath the skin, but they may be from almost any mucous surface or into any organ of the body.

Etiology

These Haemorrhages are not common and are met with more often in institutions than in private practice. Figures compiled by various hospitals show a variation in the percentage of cases encountered. The Boston Lying in Hospital reported 32 cases in 5,225 births, or 0.6 per cent. The Lying in Hospital at Prague reports 190 cases in 13,000 births, or 1.4 per cent. In the Foundling Asylum of Prague, 8 per cent. of 740 infants suffered haemorrhagic disease of the newborn.

The condition is not a manifestation of Haemophilia. Haemorrhages in the newly born are only slightly more frequent in males, while in Haemophilia males predominate 13 to 1.

Haemorrhagic disease of the newborn is self limited, and runs a definite course to recovery or death. The tendency to bleed does not extend beyond a few weeks and often lasts but a few days. Circumcision has been done within a few days after cessation of the haemorrhages without any unusual bleeding. In a case under observation with most extensive subcutaneous haemorrhages, all tendency to bleed had ceased before the separation of the cord, although there had been previously bleeding at the navel. The bleeding occurs with about equal frequency in feeble and well-nourished infants. Syphilis is associated in but a small proportion of cases.

An associated sepsis has sometimes been noted. During one year of service at the Nursing and Childs Hospital, New York, there were eight marked cases of Haemorrhage in about 225 deliv-

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ered. While more cases than usual occured among the children that year, it was striking that *not one of these haemorrhagic cases gave any evidence of sepsis*, and that none of the septic cases had bleeding. Yet the circumstances in which these haemorrhages sometimes occur point strongly to an infectious origin. The results, often remarkable, following the injection of human blood Serum indicate that the essential cause, in the largest number of cases, is a lack of some substance in the blood essential to coagulation. Sufficient studies have not yet been made to establish the precise nature of these blood changes. *The results of treatment would seem to show that the cause of these haemorrhages is not always the same.*

While the haemorrhage is not traumatic, bleeding is exceedingly prone to occur in the skin over pressure points such as the back, the elbows, the occiput and the sacrum. It is also common from the mucous membranes which are the seat of pathological process, especially the eyes, the nose and the genitals.

Morse offers an explanation of the condition based on the blood chemistry findings in non-haemorrhagic and haemorrhagic infants.

During the first four or five days of life there is a definite prolongation of the coagulation time with a gradual reduction to normal by the tenth day. That is, the normal condition in the blood in the newborn is favorable to the development of haemorrhagic disease of the newborn. If the normally prolonged coagulation time is exaggerated, the conditions are even more favorable for the development of spontaneous haemorrhage.

Haemorrhage developed, however, in only a small percentage of cases in which the coagulation time is prolonged, even if it is much prolonged. Furthermore, haemorrhage develops in babies in whom the coagulation time is not prolonged. There is much difference of opinion as to whether the bleeding time is prolonged or not, and if it is, whether it plays any part in the etiology of spontaneous haemorrhages of the newborn. If it does it is probably not the primary factor, but merely secondary or contributory.

There is a definite diminution in the prothrombin of the blood during the period in which the coagulation time is prolonged and when the prothrombin reaches the normal level the coagulation time becomes normal and the tendency to haemorrhage ceases. The calcium content of the blood of the newborn is greater than that of older children, the blood platelets are normal in number, there is no defect in the retractility of the clot and no fibrinolysis. These are exactly the conditions present in Haemophilia, which is due to a qualitative defect in the platelets.

It seems probable, therefore, that spontaneous haemorrhage in the newborn is due to a deficiency of prothrombin, which is, in turn, due to a qualitative defect in the blood platelets. It is evident that the blood platelets soon become normal and contain a normal amount of prothrombin. Why the platelets are deficient in prothrombin at birth and why they cease to be deficient after a few days is unknown.

The best method of determining the coagulation time is by means of a watch glass containing a lead shot. When the blood clots the shot will not roll.

Pathology

Haemorrhages, sometimes extensive, in various parts of the body, gastro-intestinal canal, mouth, nose, navel or the viscera, are not infrequent in the first few days of life. Aside from the occasional discovery of ulcers in the gastro-intestinal walls, the reason for these haemorrhages is not evident at autopsy. This condition has been called "Morbus Maculosis Neonatorum." Haemorrhages from the skin, mucous membranes or navel may take place in the syphilitic newborn. Haemoglobinuria may occur in epidemic form in young children.

"In very many cases, the autopsy shows nothing except the haemorrhages in the various situations and the

blanching of the organs due to the loss of blood. The haemorrhages of the brain are usually meningeal and diffuse. The pulmonary haemorrhages are small and unimportant and large haemorrhages into the pleura or pericardium are very rare. The stomach and intestines may contain considerable blood variously disorganized in the various parts of the canal and there may be ecchymoses of the mucous membrane. In addition, ulcers may be found in the stomach and the duodenum. The ulcers are multiple, small and usually superficial, but may extend to the muscular coat and may even perforate. The intestinal ulcers are found only in the duodenum and resemble those of the stomach. The cause of these ulcers is somewhat obscure, some of them are undoubtedly dependent upon inflammatory changes, probably of infectious origin; others have been compared to the peptic ulcers of later life, and are attributed to thromki in the blood vessels of the mucous membranes. These ulcers are found in but a small proportion of cases in which bleeding occurs from the alimentary tract and they may be wanting even when it has been very profuse. Small extravasations may be seen upon the surface or in the substance of any of the abdominal organs. The changes found in the blood have not been uniform.

Symptoms

The onset is frequently in the first week of life—very rarely after the twelfth day. The haemorrhages are usually multiple.

In many cases nothing is noticed until the haemorrhage begins. The first bleeding noticed may be from the stomach, intestines or any of the mucous surfaces, beneath the skin, or from the umbilicus. The amount of blood lost in most cases is not great, but there is a continuous oozing. The total haemorrhage may be only a few drams or it may reach several ounces. The general condition is one of considerable prostration, often from the onset. In all cases there is a rapid loss of weight—the temperature may be high, low or subnormal. A marked elevation of temperature may not depend upon the haemorrhage, but upon associated conditions. In a large number of cases there is diarrhoea.

The duration of the disease in cases which recover is usually but one or two days. In fatal cases it is rarely more than three days, often less than one. Death may result from the gradual failure of all the vital forces or from rapid loss of blood.

It is well to discuss somewhat in detail the more obvious clinical localities of haemorrhage, to which the physician's attention is at once drawn, during the course of his examination of the infant.

Umbilical Haemorrhage.—A slight oozing from the umbilicus not infrequently occurs when the ligature has been improperly applied. This is generally controlled by simple measures. Spontaneous haemorrhage is quite different. It occurs rather later than bleeding from the mucous membranes, usually occurring between the fourth and seventh day. There may be bleeding into the cord as well as from its far extremity. A slight stain upon the dressing is usually the first note of warning, but in exceptional circumstances a gush of blood is the first symptom. The haemorrhage may be temporarily arrested by various means, but it shows a strong tendency to occur in spite of everything that is done. The usual duration is two or three days. It has been known, however, to persist for twelve or fourteen days and it may be fatal in less than twenty-four hours from the onset.

Haemorrhage from the Stomach and Intestines.—Bleeding occurs much less frequently from the stomach than from the intestines. Gastro-intestinal haemorrhages begin, in the great majority of cases, during the first three days of life. The blood vomited is usually in dark brown masses and not very abundant; rarely is it bright red. The quantity varies from one dram to half an

ounce. Vomiting is likely to be excited by nursing. The blood discharged from the bowels is always dark coloured, usually intimately mixed with the stool —very rarely in clots. If in doubt between blood and meconium one should look for the corpuscles with the microscope. When this is not conclusive on account of the disorganization of the corpuscles, a chemical test for the haemoglobin should be made.

Concealed haemorrhage into the stomach may take place, which may be sufficient to produce death, no blood being vomited or passed by the bowels. In such cases autopsy may reveal quite a large quantity of blood, both in the stomach and intestines.

Haemorrhage from the Mouth.—The quantity of blood is rarely large, but it is here that it is often first seen. Its source may be the mucous membrane of the mouth, pharynx, oesophagus, stomach or bronchi. It may be associated with ulceration of the hard palate, with thrush, or with fissures of the lips.

Haemorrhage from the Nose.—These are infrequent and are more often due to syphilis than to other causes. They are rarely profuse, but are frequently repeated.

Subcutaneous Haemorrhages.—These often appear in places exposed to pressure, such as the sacrum, heels, occiput, or back, but may occur anywhere. In some cases these haemorrhages are very extensive—as in one recently under observation, where nearly one-third of the thorax was covered. When they occur alone or from the principal lesion, the prognosis is favourable.

Haematuria.—The urine is not only stained with blood, but sometimes contains clots. This haemorrhage may have its origin in the bladder, urethra, or kidney. Blood coming from kidney is sometimes due to uric acid infarcts, and may have nothing to do with the general haemorrhagic disease.

Haemorrhage from the Conjunctiva.—The blood usually comes in drops from between the eyelids, chiefly from the tarsal surface. It is generally preceded by conjunctivitis.

Haemorrhage from the Female Genitals.—This not infrequently occurs without haemorrhages elsewhere, and under such circumstances, is rarely serious. Of reported cases under six weeks of age, no case resulted fatally. These are not to be regarded as cases of precocious menstruation.

Prognosis.

Before the introduction of treatment with human blood serum, the haemorrhagic disease in the new-born had a very bad prognosis. No case should be looked upon as hopeless, for perfect recovery has repeatedly taken place when it seemed impossible.

Treatment.

(a) It has been suggested that the coagulation time and bleeding times ought to be determined in all new-born infants and that, when prolonged, treatment be instituted.

This procedure seems hardly necessary, however, when it is remembered that coagulation time is normally prolonged, that the line between normal and abnormal prolongation is indefinite, that only a small percentage of the infants in whom it is supposed to be abnormally prolonged have spontaneous haemorrhage and that treatment, after haemorrhage has appeared, is almost invariably successful.

The methods of treatment in order of efficiency are the injection of fresh animal or human blood serum or of whole human blood, and the transfusion of whole human blood. Animal serum is not as good as human. Their action is due to the prothrombin which they contain. This disappears in a few hours. They are useless, therefore, unless they are fresh. Old serums, such as diphtheria anti-toxin, are worse than useless because they may do harm in other ways. The dose is one or two ounces injected into the tissue, preferably of the buttocks. When whole blood is used it should be obtained by vein puncture and immediately injected into the buttocks. One or two ounces is sufficient.

N.B.—It is unnecessary to consider compatability when either serum or whole blood is injected into the tissues. In the majority of cases the injection of serum or whole blood will stop the haemorrhage.

(b) In the majority of cases the injection of serum or whole blood will stop the haemorrhage. If it does not the injection may be repeated in a few hours or a transfusion done. A single transfusion almost invariably stops the haemorrhage at once. Transfusion is therefore preferable. Many authors say that a transfusion should always be done and that it is not justifiable to inject blood, because of the possibility that the haemorrhage may recur, or that it may suddenly increase or appear in some other place, if the injection is not efficient. The injection of whole blood is much quicker and simpler than transfusion, however, and can be done by many physicians who are not competent to do a transfusion. Unless some one accustomed to doing transfusions is at hand, therefore, it is ordinarily safe to trust to the injection of whole blood, provided that the baby has not already lost a large amount of blood.

Direct transfusion is a very difficult operation with babies and is not necessary. Indirect transfusion is best performed with paraffined tubes, although if the operator is expert, a wet syringe may be used. Theoretically uncitrated blood should be preferable to vitrated blood, because the effect of citration is to prevent coagulation and the defect in haemorrhagic disease of the new born is in coagulation. Practically, citrated blood has been used by many operators with success. Two ounces of blood are sufficient to stop the haemorrhage—more should be given, of course, if there has been much loss of blood.

(c) It has been supposed that the blood of new-born infants was indeterminate as regards grouping and that it was, therefore, unnecessary to determine compatability before selecting a donor. It has been recently shown that with a sufficiently delicate technique, eighty per cent. of new-born infants can be placed in one of the four recognized groups and that one-third as many infants as adults have isohemolysins in their blood. There apparently should be some danger in doing transfusions in the new-born without determining the compatability of the blood before selecting a donor. Practically, however, the experience of many men over many years has shown that the danger is so slight that it is negligible. Nevertheless, if there is time and the facilities are at hand for making the tests, it is advisable to determine the compatability and to use only a suitable donor. If the time is limited or the facilities are not at hand, transfusion should be done with any donor available.

The administration of drugs by the mouth,of adrenalin subcutaneously or of gelatin by any means, is useless. Bleeding from the surface of the body can be sometimes temporarily diminished or stopped by the application of thromboplastin or adrenalin Constant pressure, however, usually gives better results. If the cord is bleeding it should be united, but no wounds should be made in it in retying.

Conclusion.

In preparing this paper the work of many authorities has been reviewed. Many of the findings and treatments recorded by writers of only a few years back are not wholly in accord with the latest literature. For this reason this material has been gathered from the very recent editions of work on the subject.

The majority of us have never seen a case of Haemorrhagic Disease of the Newborn. No doubt many of us will practise medicine for a life-time and never see a case, yet we must be prepared to diagnose and treat if the occasion should arise.

Those who are so fortunate to practise in a municipality where there is hospital service will have no difficulty in the treatment,—transfusion by all means. Yet we must consider the phy-

sician who is in such a position that the necessary equipment and help for treatment are not at hand—we must be ready then to use the alternative treatment—that of blood injection.

In concluding, I advise you to bear in mind the following facts:

(1) Haemorrhagic Disease of the Newborn is of obscure etiology.

(2) Its diagnosis is not difficult.

(3) It occurs during first two weeks of life—more often during the first few days.

(4) Its treatment is almost specific:

(a) Transfusion if possible 50-100 c.c. of whole blood.

(b) Blood injection 20-30 c.c. Repeat the injection if necessary. The injection is given intramuscularly into the buttocks; an ordinary 20 c.c. syringe is the only equipment necessary.

Daffydil 1928

Daffydil this year maintained the high standard established in recent years, and those who failed to put it on their list of amusements made a serious error, for laughs were almost continuous. The frolic was held on the evenings of February 16th and 17th in Convocation Hall, and the traditional banquet in the Pompeian Room of the King Edward Hotel on the Saturday evening following.

A drama of the desert entitled "Beau's Jest," written by M. J. Kelly and G. H. Hutton of the Fourth Year and presented by ten members of the same year carried off the prize. It was a rollicking skit containing several good parodies, which will probably become standard Daffydil songs.

Five recent graduates, all of them well known in Daffydil circles from past exploits, put on one of the best skits of the evening. This is an innovation which should most decidedly be made a permanency. The General Skit disclosed a somewhat alcoholic scientific discussion group in which quite a number of our most popular instructors figured more or less characteristically. The girls, as usual, added materially to the general tone of the performance and, this year, presented a quaint little scene in which a little girl's dolls came to life and made merry while she slept. Professor Klotz, Prof. McLeod, Prof. Linell, Dr. VanWyke, Dr. Cameron, and Dr. Ball were the judges of the performance.

This is the first year in which any serious attempt has been made to combine the Daffydil Banquet and Faculty Dinner, and though neither staff nor students were present en masse, it was voted a success. It is to be hoped that the idea of a Faculty Dinner, such as is an institution in many other schools will take hold, and that next year may see a more general attendance.

MEDICAL WOMEN'S HOCKEY TEAM, 1928.

The Medical Women's Hockey Team this year is comprised of members from nearly every class in the Faculty. After steady practice the girls defeated Victoria II. with a score of 2-0. In the first tilt with the University College team, neither team was able to score, but in the second, the U. C. girls were victorious—score, 1-0. They have yet to play off the tie with U. C., and the return game with Victoria II. So far there is every chance of Meds winning their group, which fact is due in great part to the careful coaching of Jack Lind. Line-up: Goal, M. Grant, K. Baldwin; defence, E. Gee, M. D. Laird; forwards, H. Craw, V. Henry; centre, Dot James; subs, M. Smith, Jean Mustard, G. Mahon.